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The lasting health and income effects of public health formation in Sweden

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Abstract

Socio-economic inequalities are remarkable in contemporary developed countries and continue to grow. The sources of these phenomena are not understood, and there is no agreement as to when in an individual's life they originate, from early childhood to adulthood. The literature showing that health in infancy may be an important factor in later-life health and income trajectories is expanding, but empirical evidence is still scarce. This paper is the first to link differences in individual access to better health care during infancy to income and health outcomes in old age. Due to the public health care reform that became one of the first elements of the Swedish welfare state, between 1890 and 1917, all rural areas established local health districts that implemented preventive measures with regard to the spread of infectious diseases. Using administrative longitudinal population data and exploiting exogenous variation in the timing of the implementation of the reform across parishes, we examine whether individuals treated in their infancy have an advantage in old age. Our findings indicate that treatment in the public health care system in infancy leads to a significant reduction in mortality, with the largest effects on cardiovascular diseases and to an increase in individual permanent incomes. The effects are universal across different subpopulations, with somewhat stronger responses among individuals from poor socio-economic backgrounds.

JEL: I14, I15, I38, J26

Key words: early-life, reform, health district, mortality, income, life-course, Sweden

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Introduction

Our knowledge of the long-term consequences of public health programmes implemented universally is extremely limited (Currie & Rossin-Slater 2015). More consistently, these programmes have been recognized to have a beneficial effect on individual and population health (Gluckman *et al.* 2008). Otherwise, the focus on both health and productivity determined by childhood health through public health programmes is a powerful way to establish the causal link from health to income and income inequalities (Weil 2013; Bleakley 2010). Using the design-based identification strategy for causal inference, the aim of this paper is to explore the effects of health in infancy on health and pension incomes in old age. We exploit the beneficial changes in health among infants, which were driven by exogenous variation due to the universal public health reform that was implemented in the rural parishes throughout Sweden for the cohorts born between 1890 and 1917. The detailed register data allow us to follow all individuals born in rural parishes, with different timing of the implementation of the reform throughout Sweden, from their 70s to death. Both pension income and mortality outcomes are available, and we therefore capture the benefits of better health in childhood in terms of quantity and quality of life. The rich supplementary dataset on the reform implementation enables us to untangle the effects of initial socio-economic characteristics and the quality of the health initiatives undertaken at the place of birth.

The case of Sweden at the turn of the nineteenth century provides an ideal setting to explore the issue. The reform subsidized the creation of public health districts throughout the country and became one of the first elements of the early welfare state, intended to provide access to public health care to the entire population. This initiative resembles the experience of all developed countries in the past and developing countries more recently. All related public initiatives focused on the prevention of the transmission of infectious diseases at the community level, making the mechanism beyond the role of these initiatives in child health clear (Engberg 2007; Mooney 2015). The establishment of rural health districts was a nationwide experiment, as up until the first quarter of the twentieth century approximately 80 per cent of children were born in rural areas in Sweden. This also means that for the cohorts treated by this reform, we discern no overlapping public health interventions, such as water system improvements, food regulations or vaccinations, which were solely targeted to cities during that period (Nelson 2002). Despite the dominance of measures focused on the prevention of disease transmission rather than water treatment, beginning in the 1880s, infant and child mortality rates dropped in Sweden as rapidly as in other countries, indicating the relative importance of preventive measures in improved life expectancy (Lazuka *et al.* 2016). Moreover, the parishes that adopted the reform programme earlier were poorer than others in terms of wealth and previous public health expenditures and were in need of a health district as a health institution. This feature is also unique to Swedish public policies because all known public health initiatives in other developed countries of Europe and North America in the past led to much quicker reform adoption among affluent communities (Cutler & Miller 2005; Kesztenbaum & Rosenthal 2014). The outcomes of the Swedish reform therefore did not emerge due to community wealth or

income, but rather can be considered as exogenous and attributed solely to improvements in child health.

Our study attempts to make important contributions to the existing literature. To date, the literature studying the link between broad early childhood conditions and later life outcomes is predominantly correlational. Causal studies of this kind employ either specific and abrupt negative conditions or universal reforms implemented in the mid-twentieth century at the earliest (Almond & Currie 2011; Currie & Rossin-Slater 2015). Our paper analyses the impact of the improved access to health care provided to the general public along with the formation of the welfare state, policies which are similar to all developed countries in the beginning of the twentieth century or in many developing countries more recently. Furthermore, the existing literature is limited to adult health as a main outcome of childhood circumstances. The findings from several studies where socio-economic outcomes are investigated are inconclusive or mixed. However, the focus on both disease and income phenomenon as determined by childhood health is probably the only way to provide the causal link from health to income and income inequalities. This paper examines both mortality and income, thereby filling this gap. Due to the recent rollout of the reforms, the follow-up period in contemporary studies is limited to young and middle adulthood (Smith 2015). Thus, the focus on old-age disease and well-being in this paper could shed light on the causes of chronic diseases and conditions that today constitute the main burden of ageing societies. Policies targeting old-age populations through contemporaneous treatments are expensive, and they could be substantially relieved by a focus on favourable early-life conditions associated with public programmes, leading to lasting effects throughout life. In the case of the reform studied in this paper, the provision of the access to health care cost no more than 0.1 per cent of national income at the time of implementation. Finally, to date, the literature has identified two sets of environmental childhood conditions, nutrition and disease, which are capable of generating lasting effects (Montez & Hayward 2011). We contribute to this literature by studying the reform that focused on the prevention of infectious diseases, with the most discernible treatment effects during infancy.

Previous research

In the literature, there is an increased concern about the widening socio-economic inequalities in contemporary developed countries. Socio-economic differences in mortality and disease increased throughout the second half of the twentieth century in the US and Western countries (Palloni *et al.* 2009, Mackenbach *et al.* 2008). Almost simultaneously, income and wage inequalities grew and continue to grow within countries (Autor 2014; Deaton 2013). This similarity between income and health parallels historical improvements in health and economic growth (Haines & Ferrie 2011). Encouraged by Kuznets (1955, 1963), scholars hypothesized the existence of a strong relation between improvements in living conditions due to the industrial revolution and income inequality. In a similar manner, the distribution of health in a society had been seen as an outcome of economic growth, where different socio-economic groups receive relatively different gains (McKeown 1976). However, it has become apparent

that such propositions are not empirically supported in the historical evolution of either income or mortality inequalities (Piketty & Saez 2003; Bengtsson & van Poppel 2011). The strong commonalities between these phenomena promoted another line of research that seeks a link between health and income and income inequalities. This research has been to a large extent inspired by Preston (1975), who related the mortality decline between the 1930s and 1960s to the advances in public health and medical science rather than to the gradual increase in income.

The long-term pathway from health to income has received attention in the macroeconomic literature. The bulk of those studies model health and income growth in a static relationship. In the neoclassical tradition, health is considered as a form of human capital, among other inputs that affect labour productivity (Bloom *et al.* 2004). In a similar manner, health improvements could additionally act as inputs to population and total factor productivity (Kalemli-Ozcan *et al.* 2000). Health and human capital are assumed to be crucial for economic growth in endogenous growth models, where they are self-perpetuating and thereby enable labour productivity to grow beyond the provided inputs (Fogel & Costa 1997; Lucas 1988, 2009; Romer 1990; de la Croix, & Licandro 1999). The co-evolution of technological knowledge and institutions, in particular those that promote public health, could also be central to long-term economic growth (Mokyr 2005; Galor 2005; Agenor & Neanidis 2011). The majority of the empirical country-level studies examining contemporaneous relations find robust reduced-form effects of health on income and more moderate effects on income per capita (Weil 2013). The emerging literature goes beyond period effects of health on income and considers its dynamic nature. The rationale is borrowed from recent studies in economic and epidemiological studies that emphasize the relatively larger benefits of health interventions for the younger segment of the population (Kuh & Ben-Schlomo 2004; Bleakley 2010). From this viewpoint, the full benefits of health improvements are realized only in the long run, not less than over half a century. The related empirical findings, which are based on simulation methods, demonstrate that the impacts of health on income per capita account for a sizable portion of their correlation (Ashraf *et al.* 2009; Young 2005).

To date, the microeconomic literature has provided well-defined reasons for the effect of health on income. From a purely economic view, the improvements in health could be seen ultimately as direct inputs to worker efficiency, both physical and mental (Grossman 1972, 2000). There are also several indirect proximate channels through which health influences income. Regarding human capital, health gains enable one to acquire more and better schooling, as well as the incentive to learn due to a longer life span (Ben-Porath 1967; Hazan 2009). Similarly, capital formation and overall productivity are fostered, as healthier workers use it more efficiently and partly as increased life prospects encourage individual saving rates (Bloom *et al.* 2003). Taking all these arguments together, in order to obtain the largest health and income benefits, health inputs should occur at a younger age. This notion has been further supported by testimonials from epidemiological studies that highlight the lifelong effects of inputs into physiological and cognitive development in early childhood (Gluckman *et al.* 2008). There is also an emerging economic literature that demonstrates that beginning in early childhood, health and human

capital formation occurs in a dynamic and self-reinforcing way, manifesting in health and income throughout life (Heckman 2007; Cunha & Heckman 2007). An additional mechanism runs through the responses of parental resources to the health improvements of children in the form of changes in fertility or within-family allocation of resources (Soares 2005; Currie 2009; Cunha *et al.* 2010). The income effects observed later in life are therefore a by-product of both environmental conditions and parental responses to the changes in child health.

In recent decades, micro-empirical studies on long-term health and income effects of changes in child health expanded rapidly. The overwhelming part of this literature examines the correlations between child health and parental socio-economic status and their development, which enabled the study of broad relationships. The evidence from many studies consistently suggests that differences in socio-economic origin strongly associate with child health status and persist as children age (Currie 2009). In a similar manner, parental socio-economic status correlates with education and wealth in working and old ages (Case & Paxson 2010). Long-term causal studies of this kind are rare and concerned with effects in the most disadvantaged groups enhanced by cash transfers, although they tend to establish a link between child health and education and income during the working ages (Aizer *et al.* 2014; Hoynes *et al.* 2016). Empirically, our knowledge of causality from early childhood and later health and income relies largely on studies using negative events in environmental conditions as identification strategies. Most consistently, micro-level studies based on infectious disease outbreaks or nutritional deprivation demonstrate a strong relationship between exposure in early childhood and health in young and old age (Myrskylä *et al.* 2013; Quaranta 2014; Currie & Almond 2011). Studies connecting variations in disease environment in early life to later-life income or socio-economic status, albeit scarcer, tend to capture the presence of the effects in the individual working ages (Kelly 2011; Bleakley 2010). The main concern in drawing conclusions from these studies arises from the disease and nutrition insults being local and abrupt, and therefore different from the pervasive long-term changes in environment surrounding birth and childhood.

Our knowledge about the long-term income and health effects of public health programmes is extremely limited. The scope of the interventions applied in the analysis ranges from those improving nutrition and the disease environment and providing general care in early childhood. Feyrer *et al.* (2013) find that cohorts treated with iodized salt in the US in the 1920s underwent beneficial effects in diseases related to cognitive development. Bleakley (2007) establishes the strong effects of improvement in infant health due to the hookworm eradication campaign in the US South on literacy and earnings. Well-child visit programmes initiated in different Scandinavian countries in the 1930s tended to generate positive lasting impacts on health and less consistently on incomes in the treated cohorts (Bhalotra *et al.* 2015; Hoynes *et al.* 2016; Hjort *et al.* 2014). Bhalotra & Venkataramani (2013) utilize the sudden arrival of sulpha therapies in 1937 to examine the impact of pneumonia in infancy on education and income outcomes during the working ages and find large impacts on each. Glied & Neidell (2010) find moderate effects on income associated with treatment by water fluoridation in childhood initiated in the 1940s in the US. Some of these studies appear to demonstrate larger benefits of

the reforms in children from poorer backgrounds, pointing to the distributional effects of public health provision at a society level. Defined more broadly than disease and nutrition, the policies in the empirical literature targeted specific diseases and as a result enabled the examination of one of the many channels between the influence of child health on later health and income. The investigation of the long-term beneficial effects of the public health reforms that were initiated during the formation of the welfare state represents a gap in the literature.

Context

In Sweden, economic development had accelerated by the last quarter of the nineteenth century. According to recent estimates, in per capita terms, the real GDP grew at an annual rate of 1 per cent prior to 1890, and throughout the twentieth century this rate was constant at 2 per cent (Schön & Krantz 2012). The last subsistence crisis occurred in Sweden in the late 1860s (Sandberg & Steckel 1997). Beginning from 1880, employment in industry increased from 15 to 35 per cent, although by the 1920s, approximately one half of the population still worked in agriculture (Statistiska Centralbyrån 1969). In the following decades, the Swedish manufacturing sector became dominant and services witnessed an expansion. The same development is observed in the real wages of workers, which began to gradually increase in 1880, and accelerated at the middle of the twentieth century (Jörberg 1972). Distinctly for Sweden, the majority of industrial workers were employed and lived in rural industrial locations. Urbanization followed a similar pattern. The urbanization rate was slow prior to 1890 and afterwards amounted to 2 per cent per year (Statistiska Centralbyrån 1999). Despite this increase, by the first half of the twentieth century, the majority of the Swedish population was still rural. According to international standards, Swedish cities were small, with the population of the biggest cities, such as Stockholm and Gothenburg, not more than 500,000, which accounted for less than one-tenth of the total population. The share of population that resided in the countryside (*land*), and therefore outside cities (*stad*) or semi-urban locations (*köping*), declined only from 80 to 70 per cent between 1890 and 1920 and to less than one-half of the total afterwards (Statistiska Centralbyrån 1999).

The improvements in population health in Sweden exhibited a similar pattern. There was no trend in change in life expectancy at birth until the middle of the nineteenth century, whereas afterwards it increased almost linearly from age 42 to 78 (Bengtsson 2006). Human stature also increased, although according to some estimates these changes were delayed to 1880 (Sandberg & Steckel 1997). The Swedish population increased rapidly, from approximately 4.2 to 5.9 million between 1880 and 1920 and doubling by the turn of the century (Statistiska Centralbyrån 1999). Prior to the first quarter of the twentieth century, such rapid population growth was largely a result of declining death rates, mainly among children and infants, from airborne and foodborne infectious diseases such as whooping cough, measles, diphtheria and diarrhoea (Preston *et al.* 1972). Between 1880 and 1920, the infant mortality rate declined dramatically, from 129 to 72 per 1000, and death rates in the 1–15 year-old population fell even more rapidly, from 13 to 4 per 1000 children (Statistiska Centralbyrån 1999). Among the

primary demographic processes contributing to the slowdown in population growth in this period, several stand out. These include the Spanish flu, which killed slightly less than one per cent of the population and scarred many more, gradually falling birth rates, and mass emigration to the United States, which subtracted approximately a million residents (Hofsten & Lundström 1976). In later decades, the improvements in life expectancy have been attributed to the decrease in death rates among the working population and the elderly. With regard to the causes of death, in this period the most dramatic reductions were witnessed in mortality rates from pneumonia, degenerative diseases of organs and tissues, and vascular and heart diseases (Preston *et al.* 1972; Statistiska Centralbyrån 2010).

The Swedish authorities recognized the need for the provision of public health in the nineteenth century. After the establishment of the national vital statistics in 1749, the medical board produced several reports about mortality in the country and underlined the necessity to prevent deaths from smallpox and other infectious diseases (Johannisson 2006). Under the need to save the labour force, in the beginning of the nineteenth century, the government set up the foundation for public health care, which introduced compulsory vaccination against smallpox and obliged the parishes to open poorhouses. Local hospitals and physicians received much less focus in these measures, although their free public offer became assured. However, with regard to the overall panorama of infectious diseases, any public measures before the 1880s remained responsive. For many years, under the fear of epidemics spreading to neighbouring countries, the authorities imposed quarantine regulations and the inspection of cargo, in addition to mobilizing medical practitioners (Bourdelais 2002). In the mid-nineteenth century, such initiatives covered only the major Swedish towns, which by that time were equipped with hospitals and medical personnel. However, the epidemics appeared to fail any broad and costly quarantine and surveillance efforts in the urban localities and killed many more in rural areas, where no measures were in place (Niemi 2007). By disentangling the mode of transmission of infectious diseases and the causal agents, the international bacteriological discoveries in the 1870–1880s helped to target public efforts and provided tools to combat disease. Consequently, the governmental authorities had to admit the necessity for a radical programme of public health care provision throughout the entire country.

1890 Health district reform

From 1890 until the 1920s, all communities in Sweden gradually received access to public health care in the form of local health districts. The institute of a provincial doctor district (*provinsialläkardistrikt*), which is organized around an assigned doctor, midwives and a hospital, dates back to 1773. Until the mid-nineteenth century, the number of centrally introduced health districts amounted to 2 per 100,000 inhabitants and disproportionally covered the more urbanized locations (Medicinalstyrelsen 1907). In 1840, the industrial elite was granted the right to organize a local medical district serving their residencies. After establishing a community council representative of all taxpayers, a few decades later, the local government reform extended this right to all parishes (Lindblom 1967). The local health administration

instructions followed shortly, which prescribed each parish or group of parishes to set up a public committee, including a magistrate and a doctor, to address public health matters, in particular in controlling the spread of infectious diseases. The location-initiated creation of health districts accelerated accordingly, beginning in the 1880s. However, without any government subsidies, between 1840 and the late 1880s, the process of expansion of provincial doctor districts was sluggish and favoured wealthy and industrialized locations (Medicinalstyrelsen 1907). Driven both by international achievements in municipal governments and medicine and by rapid industrial and population growth in the countryside, in 1890 Swedish state authorities announced a reform aimed at creating medical districts in all parts of the country, giving this opportunity equally to economically disadvantaged areas. According to the reform, each group of parishes with 8,000–12,000 inhabitants applying for an organization of a public health district could be subsidized with 1,500 SEK from the government and had to accumulate 2,500 SEK from local sources. Additionally, the state began to stimulate the graduation of young medical professionals and attract them to rural parishes, primarily by guaranteeing career promotions and public pensions. The reform therefore was designed centrally to bring access to public health care, with more local resources devoted to it in the rural populations.

Figure 1 demonstrates the progress in health care reform across Sweden and the considerable variation in timing across different regions. According to our data (see Appendix A for description), separate health district doctors were introduced gradually throughout rural parts of Sweden. There were 124 districts in addition to the 163 districts that existed prior to the expansion; 17 of them were created in 1881–1889 and 107 districts, subjected to the subsidies, in 1890–1917. Our calculations show that the objectives of the reform were realized (see Appendix B). The average health district cost the public approximately 3,000 SEK in real terms, which primarily was spent on the opening of a hospital or a medical station, and the employment of one doctor and four to five midwives. Access to health care at the community level was given to 12,000 inhabitants that resided in 6–7 parishes on average. At a minimum, the 1890 reform doubled the access of the population to public health care. Induced by the reform, public health spending increased by more than 680 SEK per 1000 inhabitants in real terms, whereas in the preceding decade the average public health spending amounted to 300 SEK per 1000 for the whole country and was lower in rural areas. With regard to personnel, parishes additionally employed 6 midwives and 2 doctors per 10,000 inhabitants, with 5 midwives and 1 doctor per 10,000 a decade prior to the reform.

[Figure 1]

Taking advantage of the plentiful parish-level data describing socio-economic, infrastructure, health and demographic domains of the parishes, we can analyse which factors were associated with the implementation of the reform. Our concern here is that the forerunning parishes had other favourable characteristics, such as wealth or health, which could foster the beneficial long-term effects instead of the reform. Our analysis does not support this conclusion (Appendix C). Instead, with regard to wealth, the parishes that established the reform earlier

were likely to be on average poorer compared to later adopting parishes. More specifically, accelerated implementation of the reform was associated with lower real total investment and investment into health care prior to the reform, larger population and its growth, and a smaller fraction of the active population in the labour force. Regarding population health, the public health reform under analysis was introduced independently of the disease conditions in the parishes, measured, for example, by the share of infants in the total population or the share of the disabled or the mortality rate under age 15. These results are fully consistent with earlier studies of this reform (Lazuka *et al.* 2016). That study examined the implementation and the contemporaneous effects of the same set of public initiatives in the rural parishes of southern Sweden in 1870–1940. A careful investigation of the public health initiatives showed that improved access to health care was driven by the nationwide fear of epidemics and the recognition of the lack of local competence in its prevention, and this does not correlate with local prosperity or actual infectious conditions. Similarly, no other health measures, such as water supply installations or food inspections, overlapped with the public health care measures.

The initiatives undertaken in the parishes due to the 1890 reform were intended to prevent the spread of infectious diseases. Due to the bacteriological discoveries in the 1870–1880s, the intervention was able to target disease in localities, even though antibiotic therapy and vaccination were adopted only in the late 1930s. The previous papers exploiting the same public health initiatives discuss the tools available to the public in the pre-drug decades and describe those which came in use due to the reform (Lazuka *et al* 2016; Lazuka 2016). The newly assigned provincial doctors became responsible for the monitoring and isolation of infected persons from the rest of the parish population. The cottage hospitals or health stations in the parishes were built for this reason, whereas chronic patients for many years were delivered for in-patient care to the neighbouring cities. Under the control of medical doctors, the organization of health districts encouraged the employment of midwives who were more qualified in disinfection techniques and modern knowledge than previously. Prior to the reform and accompanying the introduction of the disinfection instructions for childbirth in 1881, commune midwives participated in the registration of death, did not use antiseptics during birth deliveries and therefore by themselves could be carriers of disease (Pettersson-Lidbom 2014). The public health reform therefore not only increased the overall availability of midwives but also shifted the share of the employed midwives towards the competent ones. Additionally, the medical practitioners brought more supportive treatment to the population, such as surveillance and relief of the disease. Importantly for our identification strategy, beneficial treatment by the reform did not correlate with socio-economic characteristics of the parishes nor, as our earlier work shows, with regard to the socio-economic background of a new-born child. Both the access to the public health initiatives and treatment effects in infancy did not differ for children across different socio-economic groups. This was expected, as health care was provided to the public through redistribution for no or negligible cost to the recipients (Curtis 2011). The previous studies (Lazuka *et al* 2016; Lazuka 2016) found that these public health initiatives led

to more than 50 per cent decreases in under-5 mortality rates and entirely in paediatric infectious diseases.

Data

Administrative data for the outcomes

The individual-level data used in this paper comes from Swedish administrative registers. We utilized the Swedish Interdisciplinary Panel (SIP), which combines the multiple administrative registers for all individuals residing in Sweden from 1968 until 2012 tracked through unique personal identifiers. The SIP contains the county and parish of birth of the individual, which have been merged with our data on health districts (see Appendix D). In the period under analysis, the parish of birth is accurate and corresponds with the location of the mother's residence (Riksskatteverket 1989). Based on the data, to assure consistency in ages for the cohorts born between 1890 and 1917, we focused on the outcomes at ages 78–95 (see Appendix E). The population and death registers provide records for the time at risk and the date of death of the individual. In addition, the cause-of-death register gives the primary cause of death, which we further classified into several groups. Guided by the diagnostic groups suggested by the early-life epidemiological literature (e.g., Gluckman *et al.* 2008), we distinguish deaths from infectious diseases, circulatory and heart diseases, diabetes, cancer, degenerative diseases of tissues and organs, and violent and unspecified causes (see Appendix F). During the period under analysis, the registration of the causes of death was mentored by the three revisions in the international classification of death (ICD–8, ICD–9, and ICD–10), among which the long-term follow-ups are reliable and valid (Janssen & Kunst 2004; Ludvigsson *et al.* 2011).

With regard to the socio-economic outcomes, we obtained data from the income and taxation register. In the observation period, individuals were under the pension scheme introduced in 1960 that could be claimed from age 67. The scheme provided a flat basic rate (*folkpension*) with a supplementary benefit (*allmän tillägspension*) determined as a percentage of the average 15 highest paid years (Kruse 2010). Although ceilings in the payments existed, the pensions were thus higher with either a longer working period or a steeper earnings profile. The pension system also contained a widow's pension (*änkepension*), which could be paid out either until the death of the widow or remarriage, and similar benefits could be accrued for men. As this pension was rather substantial, amounting to 90 per cent of the base rate, and approximately 40 per cent of person-years in our sample were widows, we could check the robustness of our results by excluding them from the analysis. To avoid changes in the registration of different types of income occurring through the period in question, we used the total earned income (*sammanräknad förvärvsinkomst*), which for our age groups includes pension (70 per cent) and property income (23 per cent), and combined it with capital income (*inkomst av capital*, 7 per cent). We rely on the real yearly income as an average between age 78 and the year before death or age 95 as our preferred measure of permanent income. As an alternative measure, we

constructed a mean residual income, which is a mean residual of the individual's earnings from the year of birth and its squared term along with a set of register-year dummies.

Parish-of-birth data on the reform and other characteristics

Given the rollout of the health district reform, we require accurate data for the division of the parishes into health districts and its changes (see Appendix A). We collected this information from several sources. Primarily, the reports of the health board on provincial doctor districts contain detailed data on the allocation of the parishes as well as the creation dates and funding of the new districts (Medicinalstyrelsen 1907, 1939). As the passage of the establishment acts may be misleading about the timing of movements in actual policy (Engberg 2007), we verified these divisions with several sources, such as the provincial doctor reports attained from the National Archive in Sweden (Årberättelse 1893–1936), statistical yearbooks on health care (Statistiska Centralbyrån BISOS K, SOS 1880 – 1917, Statistiska Centralbyrån 1880–1917a) and public health investment (Statistiska Centralbyrån BISOS U 1874–1917). These sources provided information on the number of the medical personnel employed, such as doctors and midwives, and public spending, both in health care and in education, infrastructure and welfare. In case of investment, we gathered public investment series for each parish before and after the establishment year and aggregated them to a health district level, which allowed us to carefully determine the intervention dates. In the analysis, to avoid purely administrative changes, we therefore rely on the implementation dates accommodated with the public health investment series.

Our sample included rural (*land*) parishes only. We also excluded from the analysis the rural parishes that developed through the period into market cities (*köping* or *stad*) in order to avoid the overlap with other public health interventions, such as sewage and water supply improvements, as well as parishes that experienced several health district re-allocations or where the adoption dates were uncertain (220 out of 2353 parishes). Although the creation of the medical districts continued from 1890 until the 1940s, we stopped following the establishment of medical districts in 1917 for several reasons. Primarily, the organization of districts after 1920 became largely administrative, when several medical units established a few decades prior were merged into a larger unit with no corresponding employment of medical practitioners. The public investment series are also unavailable for the period after 1917. Additionally, the availability of medical personnel, such as midwives, began to stagnate as institutional childbirth deliveries increased gradually in rural locations. Finally, the waves of Spanish flu, which came to Sweden in 1918–1919, not only affected the subsequent cohorts but also encouraged the revision of the public control of infectious diseases.

In the individual administrative data, cohorts from 1890–1917 are not linked to their families of origin. To fill the gap in the individual's background characteristics, which is highlighted as necessary in early-life studies (Kuh and Ben-Schlomo 2004), we augmented abundant parish-level information from other national records. The Swedish decennial censuses 1880–1910 were the main sources. The counts contain data on the occupation names, their HISCO and their

status codes, which we further standardized into a historical international social class scheme allowing us to obtain a measure of socio-economic status consistent between the cohorts (HISCLASS, van Leeuwen & Maas 2011). Among the socio-economic variables at the parish level, we constructed several, such as the share of elite and industrial workers and the share of agricultural workers in the total male population ages 15–55, the share in the labour force in total for ages 15–55, the mean family size, and the share of the married in the total population ages 15–55. Based on the titles of all local occupations, we were also able to obtain a variable indicating whether the parish had a railway or water supply installation. We supplemented these variables with other demographic characteristics of the parishes, such as the size of population, the mean age of females, and the share of females, and with health characteristics, such as the share of infants or children under age 15, the share of the population older than age 55 and the share of (non)disabled persons in the total population. The latter group we complemented with information on deaths under age 15 gathered from the national death register.

Method

The establishment of public health districts can be considered as an improvement in the individual's early-life environmental conditions. Given the gradual implementation of the reform throughout Sweden in 1890-1917, we apply a difference-in-difference approach in the following form:

$$y_{ipb} = \alpha + \beta post_b \times healthdistrict_p + \eta_p + \lambda_b + \varepsilon_{ipb}, \quad (1)$$

where y_{ipb} denotes health or income outcomes for individual i born in parish p in the birth year b, and where $post_b \times healthdistrict_p$ is an indicator for the health district established in parish p in a year of birth b and remained in place in the post-treatment period. The state guaranteed the placement of a provincial doctor for at least 5 years, and the majority of the districts were kept longer (Medicinalstyrelsen 1907); we therefore consider pre- and post-treatment periods to be 5 years each. As a result, the individuals included in a sample are 5 years old or less at the reform implementation. The alternative bounds of 3 or 7 years provide qualitatively similar results (available upon request).

Given the numerous changes in demographic and economic conditions in the late-nineteenth and early twentieth centuries, this empirical strategy has several advantages. The smooth period changes, potentially affecting childhood conditions such as a rise in real wages or decline in fertility rates, in different locations are ruled out by the introduction of the year of birth fixed effects. The parish-specific differences invariant over time, such as the local wealth, climate or institutions, are also controlled for. As the health district was introduced to a group of parishes, it is possible to include group fixed effects instead, although we settled for the parish fixed effects as more demanding. The parishes that implemented the reform between 1890 and 1917 could be distinguished from those that did not implement the reform in this period. The untreated parishes could be located closer to the medical centres existing prior to 1880 and

enjoyed larger medical personnel and public health investment availability and its continuous increases in the period under analysis. Alternatively, untreated parishes established provincial districts much later, after 1917, until the completion of the reform in 1946. Our baseline sample therefore includes parishes that established a provincial doctor district at some point in time during the period under analysis. We therefore aim to estimate *ß* by comparing changes in old-age outcomes across cohorts born in parishes that initiated the establishment of the health district to changes across the same cohorts born in parishes that did not initiate the programme. In all models, to account for the location-level unobserved correlation, we clustered standard errors by the parish of birth. The models additionally introduce sex dummies to control for the sex-specific differences in survival and income trajectories.

As our empirical strategy relies on the random nature of the timing of establishment of health districts and parallel developments in the outcomes across the parishes, we used several approaches to address concerns about retention. As the treated parishes were located in different parts of the country, in order to eliminate the treatment effect from any secular trends at the level of the county of birth, in our first approach we introduce interactions between county of birth dummies and linear trends in the year of birth. Second, based on a multisource parish-level dataset described earlier and following Hoynes *et al.* (2016), we are able to control for trends in the observable pretreatment characteristics by including interactions between parish of birth characteristics, such as levels in 1880 and their changes from 1880 to 1890, and cohort dummies. In using a difference-in-differences method, one should especially be concerned that the outcomes for the treated and untreated cohorts before the reform implementation exhibited parallel trends. We first investigate this with an event study design (Figure 2); the results indicated no diverging health and income trajectories in the pre-reform years. In the models, in order to explicitly control for diverging patterns in the outcomes in the pretreatment periods across different parishes of birth, we introduce parish-specific linear (or quadratic) time trends. The reform did not affect all rural parishes in Sweden; the majority of them enjoyed the health system that existed prior to 1890. As our final approach, following Hjort *et al.* (2014), we can match each treated parish with one that remained untreated, based on the pre-1890 parish and health district characteristics.

[Figure 2]

The matching procedure, applied to achieve symmetry in the pretreatment trends, is described in detail in Appendix G. For this approach, we calculated propensity scores and applied nearest neighbour matching, in which we allowed only one control without replacement and imposed a common support restriction. Out of 2133 rural parishes, 492 parishes introduced a provincial doctor district between 1890 and 1917. As non-implementing parishes are different in many dimensions from the implementing parishes, we matched a variety of pretreatment parish and health district characteristics. The motivation for the determinants of health district implementation is a standard model of public health care utilization that involves socio-economic, infrastructure, health, demographic and health system domains (Andersen & Aday

1978; Kifmann 2005; Grossman 2000). The list of parish-level characteristics employed in the matching procedure is precisely the same as used among pretreatment characteristics and includes their levels in 1880 and the differences between 1890 and 1880. In addition, we include health-district characteristics describing pretreatment wealth and the public health system in the parishes, such as the logarithm of real investment in public health and the logarithm of real investment in education, infrastructure and welfare. As seen, the matching procedure allowed us to arrive at 432 treated and 432 matched parishes and therefore substantially enlarged the estimation sample. Using the constructed sample, we assigned implementation dates to the matched parishes based on their treated counterparts, and compared the individual outcomes applying a similar specification as previously:

$$y_{ipb} = \alpha + \beta \hat{post}_b \times healthdistrict_p + \delta \hat{post}_b + \eta_p + \lambda_b + \varepsilon_{ipb}, \quad (2)$$

where $\hat{post}_b$ denotes post-implementation years for both treated and matched parishes. Both the treated and matched parish receive the same parish identifier, and the method therefore should be seen as a within-parish-pair comparison, where the treated parish is actually treated and the matched one is not. Given the strictness of the method, the early-life processes should be therefore extremely strong to confirm the presence of the effects.

To examine the effects of health district reform on socio-economic inequality, we estimated the heterogeneous effects. The previous research suggested that the investigation of factors of inequalities can be made in a triple difference framework (Bitler *et al.* 2014). We applied this method by introducing additional terms into eq. 1, such as $post_b \times healthdistrict_p \times subgroup_s$, which is an indicator for the health district established in parish p in the year of birth b and subgroup of parishes s, subgroup fixed effects, and the interaction between subgroups (at the parish of birth) and year of birth. The focus is on the estimate of the triple interaction terms, which should provide an indication of whether individuals from particular socio-economic groups diverged in their outcomes from the rest of the treated population. As discussed previously, our socio-economic measures are time varying and defined at a parish-of-birth level. Additionally, health-district data for public investment and the number of employed midwives and doctors enable us to relate the early-life effects to the amount of the public resources actually spent by parishes on the establishment of the district.

The specificities of our estimation sample preclude the use of particular estimators. As we analyse old-age mortality between ages 78 and 95, we apply the procedures to capture the non-linearity of the mortality rates. The simplest way to address the issue is to leave the baseline hazard unspecified by applying a Cox proportional hazard model (Cox 1972). Across all specifications, tests based on Schoenfeld residuals revealed no violation in the proportionality of the hazards. The Gompertz regression, where hazard rates can be specified as monotonically increasing over time, has been shown to describe old-age mortality rather well (Thatcher *et al.* 1998). All duration models are adjusted for left-truncation at age 78. To assure similarity with previous research, we also estimated linear probability models based on whether or not the

individual was deceased from the beginning of observation at age 78 with different follow-ups, such as age 78 plus 5 years or 10 years, as eventually these proportions should converge with the completion of lives. For cause-specific mortality, Cox proportional hazards models were also applied. We also utilized logarithms of the survival time as an outcome. Regarding the permanent income, similar concerns are addressed. To provide a comparison between the cohorts with different earnings profiles and to impede their disproportionate influence on the results, the logarithm of income is used as an outcome in linear least squares models. As the individuals are progressively dying over time, we calculated estimates by using the mean yearly income for different age ranges, such as the full follow-up period for ages 78 and 95, and the shorter ones for age 78 plus 5 years and age 78 plus 10 years. As an alternative, we used the mean residual income, which reduces the variation from measurement errors, and therefore artificial income changes compared to other permanent income measures (Lindahl *et al.* 2015).

Table 1 presents descriptive statistics for our estimation samples. Conditional on survival to age 78, the individuals in our samples die at a mean age of 86–87. This characteristic of our sample does not deviate from the actual life expectancy at age 78 for the same cohorts in Sweden in total, which is equal to 8.5 years (Human Mortality Database 2016). More than 90 per cent of individuals die within the age range 78–95, with the largest fraction due to cardiovascular diseases at 57 per cent, followed by cancer at 16 per cent, degenerative diseases at 13 per cent, and infectious diseases at 9 per cent. The share of males in a sample is approximately 45 per cent, which is expected due to their lower survival to old age compared to females. The fraction of individuals treated by the reform is slightly more than one-half. By design, we followed cohorts born 5 years prior and after the establishment of health districts and those treated who might be more likely to survive to the beginning of the follow-up. The logarithm of the permanent income in our estimation samples is 7.7 units, which aligns well with the same cohort measure for the total population (calculated from SIP 2016). With regard to both health and income outcomes and treatment variables, baseline (implemented parishes of birth) and constructed (implemented and matched parishes of birth) samples are similar.

[Table 1]

Results

Table 2 presents results for the long-term effects on mortality from the Cox proportional hazards model in a fashion of specifications discussed previously. The estimates are presented in the exponential form and therefore can be interpreted as a percentage change in the mortality risk due to the introduction of the health district in a parish. The results show that the treated individuals born within 5 years after the establishment of the health district are significantly less likely to die between 78 and 95. More particularly, they exhibit a reduction of approximately 6 per cent in mortality risk. Our estimates keep their size and robustness in

different specifications, such as those including the year of birth linear trends across counties of birth, parishes of birth and pretreatment characteristics varying across cohorts. In the matched sample, the size of the coefficient drops to a 4.2 per cent decrease in mortality risk, albeit attaining statistical significance. We additionally estimate the reform effects on mortality by using different estimators (see Appendix H). They all support the presence of the beneficial effects of the reform on mortality. The estimates from the parametric proportional hazard model, the Gompertz model, similarly suggest an effect on the risk of dying in the range of 4 and 6 per cent. Additional models indicate that the effects are present with regard to both the incidence of death and the duration until death. Followed between ages 78 and 82, the share of the deceased among the treated individuals are approximately 2 percentage points lower compared to the rest, which at the mean dependent variable of approximately 27 per cent implies a 7 per cent reduction. As expected, this effect on the probability of death significantly reduces with the competition of lives by age 95. With regard to overall duration, the treated groups of individuals again experience an approximately 7 per cent longer life until death, which in absolute terms with the mean duration time of 9.4 years is equivalent to 0.7 more years alive.

[Table 2]

To disentangle the biological mechanisms that suspend the development of chronic diseases, we looked at the estimates for cause-specific mortality in Table 3. We perform the analysis for the sample of implementing parishes and the matched sample. The long-term effects on mortality risk from the health care reform are found predominantly in deaths from cardiovascular diseases, mainly acute myocardial infarction and chronic ischaemic heart diseases, which contributed the most overwhelmingly to the general mortality, at 57 per cent of all deaths. Dependent on the specification, the individuals treated by the reform have a 5 to 6 per cent lower chance of dying from circulatory or heart diseases. In both baseline and matched samples, the decrease in infectious disease mortality is also substantial due to the reform, between 4 and 10 per cent, albeit not statistically significant. The results suggest that the reform had a substantial impact on cancer mortality risk, decreasing approximately 8–10 per cent, although it is statistically significant only in the matched sample. The estimates in other diagnostic groups, such as diabetes, degenerative diseases of organs and tissues and accidental or ill-defined causes, are not statistically significant.

[Table 3]

The estimates for income are presented in Table 4. Across all models, we used the logarithm of income, and the results are thus interpreted as the percentage changes. The estimates suggest the positive effects of the health care reform on individual permanent incomes. With the full follow-up period at ages 78 and 95, the treated individuals have a 2 per cent higher permanent income compared to their counterparts. The estimates are stable within a range of 1.7 and 2.5 per cent across different specifications, including those with parish-specific year of birth trends or exact matching. Because individuals are progressively dying at the beginning of observation

with age, we checked whether shorter follow-up periods affected our results. The subsequent models suggest effects of similar or slightly larger magnitude (see Appendix I). Similar to the mean income, the use of a mean residual income supports the previous results. More specifically, the individuals born within 5 years after the launch of the health care reform in a parish exhibit an approximately 2 per cent increase in their life-time incomes. Additionally, we excluded person-years under widowhood from the calculation of the mean income, and the results slightly increased in magnitude to 2.5–3.0 per cent. We refrained from presenting the income effects in absolute terms, as the earnings across cohorts under analysis vary considerably.

[Table 4]

Table 5 distinguishes the health and income effects by subgroups based on the quality of the public health initiatives undertaken due to the reform. As the introduction of the health district in the parishes implied the necessary implementation of the set of initiatives described earlier, the early-life effects should inevitably emerge at this extensive margin. Improving the intensive domain of the health district with more midwives employed or larger hospital facilities produced additional benefits. The estimates for the heterogeneous effects, where the implementing parishes are divided into two groups by the public health investment and additional midwives employed per parish population at the median, are supportive of this result. The effects in mortality risks are present in groups with low and high investment and are larger for the latter, at 5 and 7 per cent correspondingly. This pattern is less evident for the income effects, with lower responses to more public health spending and higher responses to a higher availability of midwives, where probably both direct and indirect channels from early-life treatment operated. The long-term effects are also related to the disease burden in the parishes, measured with the share of infants in the parish population and mortality rates under age 15. In both cases, the health effects are observed for all parishes but are larger at higher baseline levels of disease. Again, the divergence by these subgroups does not emerge for the permanent income effects.

[Table 5]

To address the role of treatment in inequality development, we present the effects of the introduction of the health district by subgroups with different socio-economic characteristics at birth in Table 6. As discussed, we consider several measures of socio-economic status in the parish of birth obtained from the decennial censuses. The estimates for both mortality and log income suggest that the treated individuals from all socio-economic backgrounds benefited from the reform, whereas the benefits are greater for the more disadvantaged groups. In the case of mortality, the affected individuals with wealthier and poorer socio-economic origins obtained 3 and 10 per cent decreases in all-cause mortality risks, accordingly. The availability of a railway can serve as a measure of parish infrastructure and communications, and here the effects on mortality are to a larger extent concentrated among the poorer parishes. Initial socio-economic conditions were stressed as an important mediator of the early-life effects, especially

for income outcomes (Flores & Kalwij 2014; Currie 2009), and here the pattern towards individuals with more disadvantaged childhood conditions is also obvious. Depending on the measure, the treated individuals of lower socio-economic status in childhood attain 3–4 per cent, and the increases among the initially richer individuals are twice as low. Similar differences in income outcomes are observed between the treated individuals born in parishes with no access to the railway. In using the parish-level characteristics to build the measures of socio-economic status in infancy, we cannot entirely distinguish whether the individuals from poor families benefited more from the introduction of the health district or whether those born in parishes with more poor families in the total population benefited more.

[Table 6]

Robustness Analyses

So far, the treatment effects for both old age mortality and income appear to have been robust to the inclusion into the models of different controls for the pretreatment secular trends specific for location of birth. They hold for the additional interactions between year of birth trends and county of birth dummies, parish of birth dummies, and level and trend changes in a set of pre-intervention observable parish of birth characteristics. In the baseline and matched sample, where the pretreatment trends are equalized based on these characteristics by the virtue of the matching procedure, the significant effects remain. One concern could still be raised in which coinciding social spending occurring alongside the introduction of health districts could explain the results instead. An additional concern is whether any discontinuous changes in specific locations coincided with the timing of the health care reform, thereby changing the composition of the individuals' parents towards wealthier or healthier ones. Finally, it is important to test whether the reform by itself affected the composition of the parents. In what follows, we analyse the robustness of our results to the potential influence of these factors.

One concern relates to selective mortality and its potential effects in our results. It is obvious that we examined the group of individuals in their old age, between 78 and 95, where selective processes could cause some cohort differences to change. This methodological issue was specifically addressed by recent studies of similar cohorts (Zajacova & Burgard 2013; Cutler & Lleras-Muney 2010) and most studies of long-term outcomes (Almond 2006; Bhalotra & Venkataramani 2013), and suggest that our results might be underestimated. More specifically, the individuals untreated by the health care reform that survived to their 70s may be a more selected group, with better health and earnings, than the treated individuals who survived to that age. In support of this, we directly assess the magnitude of the survival bias (see Appendix K). Because we have the data for the number of infants at the parish level for each cohort (from censuses) and the number of individuals who survived to age 78 (from SIP), we estimated the impact of the reform on the fraction of old-age survivors. The results show a significant increase in the fraction of survivors as a result of the reform at 7 per cent of the mean. We can further apply a two-stage Heckman selection procedure to analyse whether a selection to survival

affects our results (Heckman 1979). In the first stage, the probability of being observed in the estimation sample is modelled as a function of cohort fixed effects, county of birth fixed effects and sex for all individuals whom we observe in the year 1960 (1960 Census) in a probit model. An inverse Mills' ratio originating for each individual from the estimates of the probit model is further included as a covariate into the baseline specification, and this procedure does not affect our results (see Table 7 Panel A). The medical treatments for certain diseases, such as antibiotics and drugs for cardiovascular and heart diseases and diabetes, which emerged as contemporaneous events, should not affect our results.

[Table 7]

Another methodological issue relates to the income variable that we utilized. It can be questioned whether a mean income in old age can approximate for permanent income, that is, income in early and middle adulthood. Previously, we discussed that the pension schemes, covering the cohorts under analysis, relied substantially on the economic performance in adulthood, albeit not in full. While we believe that the use of income in old age as a permanent income leads to the underestimation of the treatment effects, it can also be viewed a measure of an individual's economic well-being (Netuveli *et al.* 2006).

Given the rise in overall social spending starting from the mid-1880s, one might question whether the effects captured emerge entirely due to the health care reform and not due to other overlapping public initiatives. We have already discussed that the local governments put in place no other public health measures, such as improvements in water facilities, vaccinations or food hygiene control, until the 1930s. From our dataset, we observe that increases in public health investment associated with the introduction of health districts also implied increases in overall social spending. A more careful look suggests that the overwhelming part of the residual local investment was spent on primary schools, which do not directly affect infants or health under age 5. Additional resources were also accumulated for poor relief, although they covered only 1.2 per cent of children under age 15 with direct support (Statistiska Centralbyrån BISOS U 1890–1917). The rise in social expenditures other than public health care is therefore not responsible for our results.

In the period under analysis, it is possible that some abrupt changes affected parishes differently, thus potentially harming our identification strategy. One potential threat to our estimates arises from the mass emigration of Swedish residents to the US and other countries, which was discontinuous in the period in question. If the emigrating population was selective towards poorer socio-economic classes, thus affecting families of our cohorts, and occurred in a sharp manner across different regions, one might expect the disturbance in our estimates. The emigration to other countries from Sweden was massive between 1880 and 1910, and approximately 80 per cent of all migrants left for the US (Statistiska Centralbyrån 1967 p. 47). Regarding the age composition of the migrants, ages 15 to 29 predominated. The primary emigrant counties, where the countryside experienced mass migration to the US, were Värmland and Halland (Bohlin & Eurenius 2010). Panel B of Table 7 presents the results for

the sample, where we excluded the individuals from the counties of birth mostly affected by emigration to the US. As seen, our results are unaffected by this exclusion. Furthermore, the location-specific influence of World War I might be important for our estimates. Even though Sweden was neutral during the war, the treated parishes could be those mostly affected by its threat, forcing the parents to postpone fertility until peaceful times, or those agricultural regions that witnessed an increase in the exports of the raw materials to Europe, thereby boosting the local wages (Siney 1975; Qvarnström 2014). We therefore ran the analysis omitting from the sample individuals born in Norrbotten and Västerbotten counties, the most affected by the war, and present the estimates in panel C. Again, both long-term income and mortality effects attain statistical significance and sizes analogous to the baseline coefficients. To supplement these analyses, we added to the models the parish of birth characteristics that more carefully describe the age structure of the population and vary across cohorts, in addition to those included previously. As panel D shows, our results were also not sensitive to this check.

We performed additional robustness analyses with regard to whether the parents responded to the reform as such. It might be possible that families, hoping to improve the life chances of their new-borns, moved to areas with access to health care and had some advantageous characteristics that could instead explain our results. The internal migration flows in rural areas in the period under analysis were explained by the structure of the local labour force (Enflo *et al.* 2014). Previously, we found that the implementation of health care reform did not correlate with shares of industrial or agricultural workers in total or the share of married persons in total. To account for residential selection, we repeated our analysis by additionally controlling for several time-varying indicators of the local labour markets, such as the share of industrial and agricultural workers in the total male population aged 15–55, the share of skilled workers in the total male population aged 15 – 55, the share of the married in the total population aged 15–55, the mean age of females, and the share of the population of non-Swedish origin. Panel E reports the results from the models including these controls, which appear to be unaffected compared to the baseline estimates. We additionally analysed the reform-driven migration responses by using the parish-level information on migrant structure obtained from censuses dated 1880–1910 (see Appendix L, Model 1). The results indicate that there were no effects of the reform on migration flow between the parishes. Finally, it is plausible that the reform generated fertility responses favouring the delivery of healthy new-borns. Again, we can test this with parish-level data from censuses (see Appendix L, Model 2); our results reveal no presence of these responses.

One could also be curious as to what extent the Spanish flu affected our results. The Spanish flu arrived in 1918–1919 and therefore affected the health and income trajectories *after* the treatment by health care considered in this study, in childhood and prime working ages of the individuals. Therefore, this death and morbidity shock does not lead to selection bias in our treatment estimates, which instead demonstrate the total effects of the reform on income and mortality, but it could mediate these effects. Previous studies of Sweden found strong immediate effects of the Spanish flu pandemic on mortality (Åman 1990), but no effects on

earnings (Karlsson *et al.* 2014). The previous literature is also consistent in that the lasting health effects from the influenza pandemic emerge only for individuals exposed in utero (Bengtsson & Helgertz 2016). In our analysis, we ran the models while excluding the individuals residing in the counties mostly hit by the Spanish flu, such as Jämtland and Västernorrland and all northern counties (Engberg 2009). As panel F in Table 7 shows, the estimates for both mortality and income are not affected by their exclusion.

Conclusions and discussion

To date, our knowledge of the long-term influence of the public health care reforms that were initiated across the developed countries during the formation of the welfare state is extremely scarce. The focus on individuals in their old age, and therefore on the prolongation of lives, ultimate incomes, and their development across socio-economic groups, disentangles the quantity and quality benefits of any such programmes (Weil 2014; Deaton 2010). Taking a causal approach, this paper examined the long-term effects of the universal expansion of health districts in rural areas of Sweden between 1890 and 1917 on income and health at age 78–95. The differences-in-differences approach assured that we compared the outcomes of the individuals born within 5 years after the establishment of the health district in the parishes with the same cohorts in other parishes that did not establish a district. We found effects for both old-age mortality and income. Due to the reform, individuals attain decreases in all-cause mortality risk at approximately 5–7 per cent, equivalent to 0.7 additional years spent alive. The positive effects on the permanent income of the treated individuals amount to 2–3 per cent. These results passed all robustness analyses, including those controlling for the secular cohort trends specific to locations and those examining the plausible compositional differences between the treated and untreated cohorts. In part, the robustness results suggest that the effect of the reform on both mortality and income are underestimated.

The estimates presented above represent the intention-to-treat estimates, and we did not directly observe the probability of the individual to be treated by the reform. In our models, all individuals born in the parishes where a provincial doctor district was opened in the post-treatment period are considered as treated, and the effects are therefore larger if any smaller proportion of them was. In our earlier work (Lazuka *et al.* 2016; Lazuka 2016), we studied the same intervention in a similar rural setting in southern Sweden that provided a detailed record of participation rates. With more qualified midwives employed in the parishes in 1890–1920, the individual-level probability of being treated by a more competent midwife compared to that by a traditional midwife amounted to 20 per cent. For the total population prior to 1880, including rural and urban areas, the proportion of licensed midwives amounted to not more than 50 per cent (Statistiska Centralbyrån BISOS K 1889–1920). With regard to other preventive initiatives undertaken after the reform, for instance, careful monitoring and forced isolation of infected persons to hospitals by the provincial doctors, equivalent likelihoods are more difficult to measure. Our previous analysis shows that the disease environment, mainly airborne infectious diseases, improved significantly due to these initiatives. As the effects for the

majority of the population occur at a group level due to the decrease in infectiousness and not due to the individual placement into the isolation hospital, the initial disease panorama, such as the prevalence of airborne or foodborne diseases, weighed the treatment probability. We demonstrated that only 46 per cent of infants treated at the location level reacted to the isolation initiatives. Therefore, to transform our intention-to-treat estimates for mortality and income into the treatment effects on the treated, the former should be divided by the proportion in a range of 0.2 to 0.5.

To the best of our knowledge, our study is the first to find effects of the health care reform on both individual health and income in old age. We can briefly align the findings of policy-based studies focusing on age and cohort groups similar or overlapping with ours. Based on the cash transfer programme initiated in the US in 1911–1935, Aizer *et al.* (2014) estimates the treatment effects on individuals accepted to the programme at ages 80 and older are approximately 1.4 additional years. Bhalotra *et al.* (2015) studies the long-term health effects of the well-child visit programme implemented in 1931–1933 Sweden, and demonstrates a 7 per cent reduction in mortality between ages 40 - 70 among the cohorts treated in a parish during infancy. Similar individual-level studies for long-term income effects are difficult to find. Albeit derived for the cohorts younger than ours, using the well-child visit programme in Norway in 1936–1955, Bütikofer *et al.* (2014) finds that the programme had intention-to-treat effects on earnings in young and late adulthood in the range of 1–2 per cent. Applying a simulation-based method to health improvements in the first half of the twentieth century and its dynamic demographic consequences, Ashraf *et al.* (2009) find that long-run effects on income per capita stabilize at an additional 15 per cent after 80 and more years. Broadly speaking, our effects on health and income are therefore within the scope of those demonstrated in the previous studies.

Our findings suggest that particular biological mechanisms can underlie the long-term effects. In the previous discussion, we highlighted that the establishment of a health district led to improvement in the prevention of infectious diseases in the affected cohorts. In this paper, the years of the establishment of the districts are used as the approximation of the starting dates of their functioning. As a result, individuals could be treated by the reform in pre- or postnatal periods or both. The event study analysis shows that the health and income effect of the reform does not differ among the affected cohorts or, to put it differently, there is the effect from the exposure in infancy and no additional effects of the exposure at ages older than one to five. The epidemiological literature emphasized infancy as most important in lasting responses to the influence of infections (Gluckman *et al.* 2008). More specifically, the body produces stress hormones or launches chronic immune responses that mobilize and withdraw nutrients away from body and brain development, thereby gradually affecting mental and physical health later in life (Danese & McEwen 2012). Additionally, infectious diseases in early life can directly damage organs and cells, leaving an individual scarred for the rest of their life (Liuba 2003). Through different channels, either improvements in productivity or human capital accumulation, health responses translate into acquired incomes (Heckman 2007). Complementarily, the human body programmed for a particular environment, once faced with

a new environment, can be maladaptive in the long run and thus generate various health, behaviour and socio-economic outcomes (Boyce & Ellis 2005). Consistent with our results, indicators of infection and inflammation are related to chronic pulmonary disease, vascular and heart disease and other diseases of ageing (Barker 1991; Finch & Crimmins 2004).

Our results indicate that implementation of the health care reform, through improved child health, led to larger effects for individuals with poorer socio-economic backgrounds. In line with our expectations, given the design of the health care reform, we find that the local preventive initiatives generated long-term benefits across all subpopulations. If socio-economic differences in health and incomes persist throughout life (Hayward & Gorman 2004; Currie & Schwandt 2016; Case *et al.* 2002), one should discern a stronger effect for the less affluent individuals. Our results provide several indications in support of this notion. Primarily, in relative terms, individuals from lower socio-economic classes enjoy larger effects in both longevity and permanent incomes. Similarly, in absolute terms, more generous public health investments per capita, devoted to the creation and functioning of the health district, generated stronger individual responses. Previously, we also discussed that the utilization of the health services occurred in an equal manner across families with different socio-economic characteristics. This body of evidence offers some evidence on the intergenerational transmission of health, where child health is linked to adult and old age health and well-being.

Our findings provide policy recommendations that could substantially relieve the burden of costs related to the support of the ageing population. Currently, chronic disease is responsible for the majority of the deaths in developed countries and globally, among which cardiovascular disease is the leading cause of death (WHO 2014). The treatment of chronic diseases and recovery and the costs targeting adulthood behaviour are extremely costly. The accent on those early-life conditions that have lasting health and income effects over the life course could reduce these contemporary costs. In most conservative terms, the public costs of the reform, studied in this paper, including continuous expenditures on health care maintenance in parishes not affected by the reforms, constitute not more than 0.1 per cent of the national income annually between 1890 and 1917 (our calculations). The health district reform was designed to provide all parts of Sweden with access to public health care, giving this opportunity equally to the disadvantaged regions, and became one of the first elements of the modern welfare state. In our previous work, we showed that the immediate decreases in infant and child mortality and, as a result, social returns to a corresponding set of public initiatives, focusing on prevention of the infectious disease from transmission, were large (Lazuka *et al.* 2016). This study provides evidence for additional gains in old age in terms of longer lives, lower probability of death from cardiovascular disease and higher permanent incomes for the cohorts affected by the reform in infancy.

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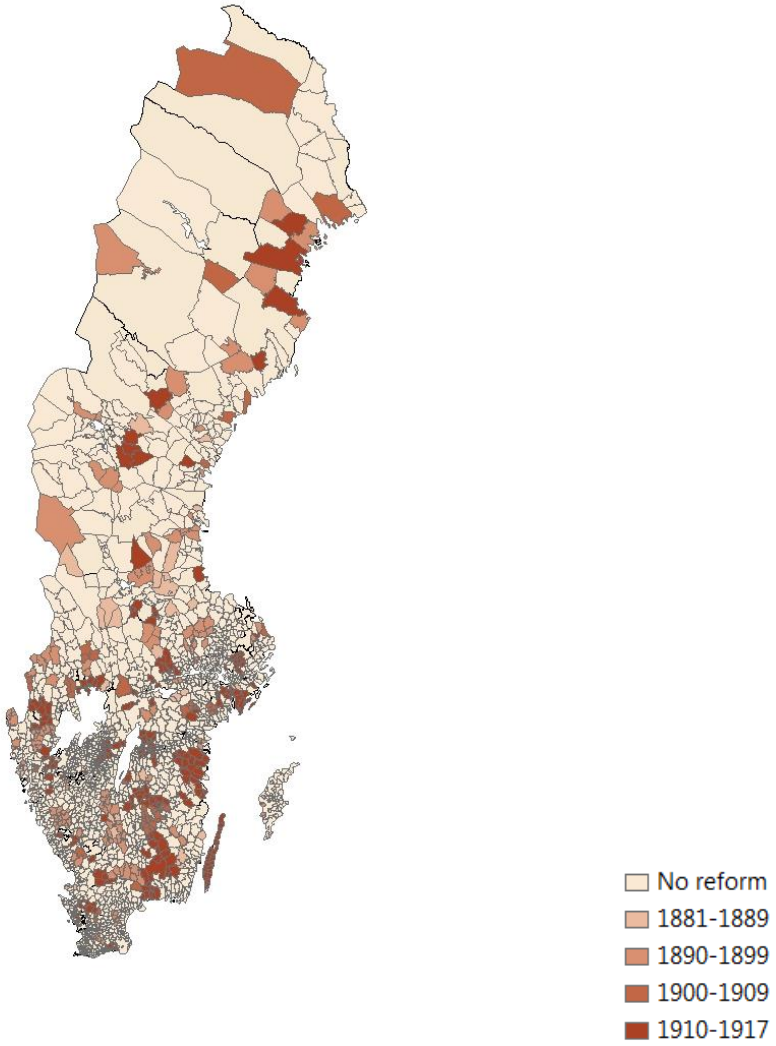
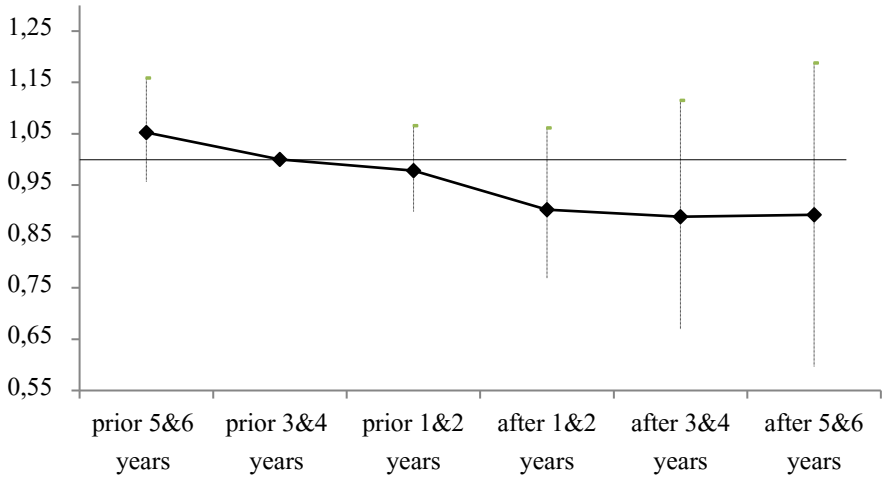
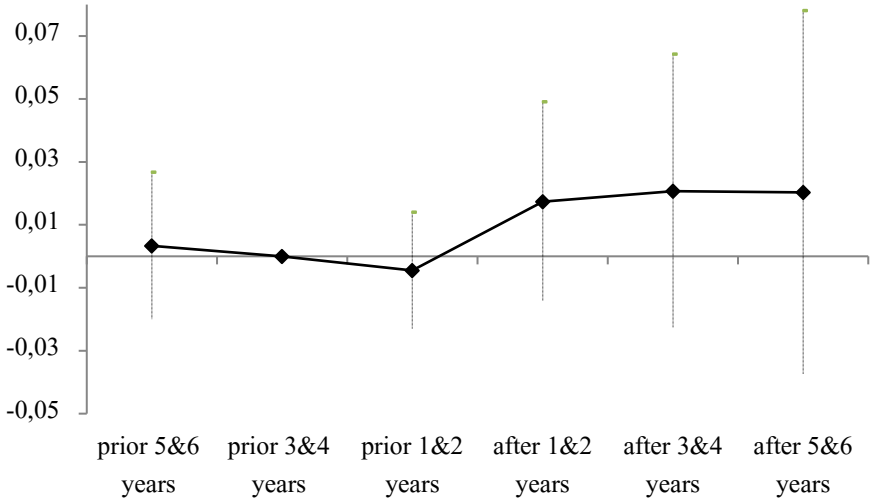


Figure 1 – Implementation of the 1890 Health district reform in Sweden
Source: Historical GIS maps from the Swedish National Archives merged with data on reform dates (see Appendix A)



(a) Mortality risk



(b) Log income

Figure 2 – Coefficient estimates before and after the reform

Table 1 – Descriptive statistics of the estimation samples

	Sample of implemented (*I*)	**Sample of implemented and matched (*M*)**
Mortality sample, ages 78 - 95		
post X health district	0.540	0.254
	(0.498)	(0.435)
mean death age (non-censored)	85.85	85.81
	(4.799)	(4.805)
mean censoring age	86.55	86.50
	(5.220)	(5.224)
fraction of observed deaths	0.920	0.922
	(0.272)	(0.269)
due to infectious diseases	0.091	0.091
	(0.287)	(0.288)
due to cardiovascular diseases	0.569	0.568
	(0.495)	(0.495)
due to diabetes	0.018	0.018
	(0.132)	(0.134)
due to cancer	0.156	0.155
	(0.363)	(0.362)
due to degenerative diseases	0.133	0.135
	(0.340)	(0.342)
due to other causes	0.034	0.033
	(0.180)	(0.179)
male	0.451	0.448
	(0.498)	(0.497)
Number of individuals	39,604	69,939
Permanent income sample, ages 78 - 95		
post X health district	0.538	0.253
	(0.499)	(0.434)
log income, mean yearly (including 0s)	7.707	7.704
	(0.551)	(0.536)
male	0.448	0.445
	(0.497)	(0.497)
Number of individuals	38,618	68,224

Note: means of the variables and standard deviations (in parentheses). Data from Swedish Interdisciplinary Panel

Table 2 – Hazard ratios. Effect of the reform on mortality in ages 78–95, cohorts 1890–1917 Sweden

	I	***I***	***I***	***I***	***M***
post X health district	0.940***	0.941***	0.940**	0.948**	0.958**
p-value	(0.006)	(0.007)	(0.035)	(0.022)	(0.012)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	39,604	39,604	39,604	39,604	69,939
Deaths	36,429	36,429	36,429	36,429	64,451

Note: exponentiated coefficients from Cox proportional hazards models. Models are adjusted for the left-truncation at age 78. *I* denotes a sample of implemented parishes of birth, *M* - sample of implemented and matched. *Parish of birth Xs* denote parish-level pre-treatment control variables and include levels in 1880 and differences 1890-1880 of the following variables: log of total population, share of elite and industrial workers in male population 15-55 ages, share of agricultural workers in male population 15-55 ages, mean age of female, share of females in total population, share of population in labour force 15-55 ages, share of married among population 15-55 ages, share of infants in total population, share individuals older age 55 in total population, mortality rate under age 15, share of disabled in total population, mean family size, whether a parishes had a railway, whether a parish had water installations. Standard errors clustered at the parish of birth level (414 parishes).
P-values in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 3 – Hazard ratios. Effect of the reform on cause-specific mortality in ages 78–95, cohorts 1890–1917 Sweden

	Infectious	**Cardiovasc.**	**Diabetes**	**Cancer**	**Degener.**	**Other**
I						
post X health district	0.899	0.939**	0.946	0.922	1.003	0.930
p-value	(0.151)	(0.035)	(0.693)	(0.105)	(0.963)	(0.589)
Cohort FE	yes	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes	yes
Individuals	39,604	39,604	39,604	39,604	39,604	39,604
Deaths	3,304	20,733	649	5,675	4,847	1,221
M						
post X health district	0.961	0.953**	0.969	0.898**	1.030	1.058
p-value	(0.454)	(0.034)	(0.772)	(0.010)	(0.512)	(0.571)
Cohort FE	yes	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes	yes
Individuals	69,939	69,939	69,939	69,939	69,939	69,939
Deaths	5,862	36,579	1,185	9,961	8,723	2,141

Note: exponentiated coefficients from Cox proportional hazard models. Models are adjusted for the left-truncation at age 78. *I* denotes a sample of implemented parishes of birth, *M* - sample of implemented and matched. Standard errors clustered at the parish of birth level (414 parishes). See main text for further description.
P-values in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 4 – OLS estimates. Effect of the reform on log income in ages 78–95, cohorts 1890–1917 Sweden

	I	***I***	***I***	***I***	***M***
post X health district	0.0203**	0.0212**	0.0228**	0.0246**	0.0170**
p-value	(0.041)	(0.042)	(0.028)	(0.033)	(0.025)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,618	38,618	38,618	38,618	68,224

Note: OLS regression estimates. *I* denotes the sample of implemented parishes of birth, *M* sample of implemented and matched. *Parish of birth Xs* denote parish-level pre-treatment control variables and include levels in 1880 and differences 1890-1880 of the following variables: log of total population, share of elite and industrial workers in male population 15-55 ages, share of agricultural workers in male population 15-55 ages, mean age of female, share of females in total population, share of population in labour force 15-55 ages, share of married among population 15-55 ages, share of infants in total population, share individuals older age 55 in total population, mortality rate under age 15, share of disabled in total population, mean family size, whether a parishes had a railway, whether a parish had water installations. Standard errors clustered at the parish of birth level (414 parishes).
P-values in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 5 – Effects of the reform on mortality and log income by quality of the reform, ages 78–95, cohorts 1890–1917 Sweden

	Mortality risk	**Log income**
Investments into health care per capita		
post X health district X low	0.949**	0.0275**
	(0.048)	(0.034)
post X health district X high	0.935***	0.0158
	(0.004)	(0.109)
Midwives employed per capita		
post X health district X low	0.951**	0.0167
	(0.045)	(0.163)
post X health district X high	0.931***	0.0234**
	(0.003)	(0.024)
Share of infants		
post X health district X large	0.896***	0.0195
	(0.001)	(0.225)
post X health district X small	0.973	0.0247*
	(0.353)	(0.063)
Mortality rate under age 15		
post X health district X high	0.923**	0.0097
	(0.015)	(0.431)
post X health district X low	0.963	0.0107
	(0.216)	(0.411)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Parish of birth X x cohort FE	yes	yes
Parish of birth X x parish of birth FE	yes	yes
Individuals	39,604	38,618
Deaths	36,429	

Note: exponentiated coefficients from Cox proportional hazards models for mortality, adjusted for left-truncation at age 78, and OLS regression estimates for log income. The background characteristics are at the level of parish of birth. All groups divided at the median. Standard errors clustered at the parish of birth level (414 parishes).
P-values in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 6 – Effects of the reform on mortality and log income by socio-economic parish-of-birth characteristics, ages 78–95, cohorts 1890–1917 Sweden

	Mortality risk	**Log income**
SES, share of skilled		
post X health district X low	0.900***	0.0297
	(0.005)	(0.135)
post X health district X high	0.972	0.016
	(0.331)	(0.103)
SES, share in labour force		
post X health district X low	0.896***	0.0431**
	(0.002)	(0.029)
post X health district X high	0.976	0.0049
	(0.400)	(0.637)
Mean family size		
post X health district X large	0.932**	0.0187
	(0.027)	(0.200)
post X health district X small	0.941**	0.0180
	(0.049)	(0.179)
Railway		
post X health district X no	0.886***	0.0371**
	(0.004)	(0.049)
post X health district X yes	0.970	0.0112
	(0.250)	(0.297)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Parish of birth X x cohort FE	yes	yes
Parish of birth X x parish of birth FE	yes	yes
Individuals	39,604	38,618
Deaths	36,429	

Note: exponentiated coefficients from Cox proportional hazards models for mortality, adjusted for left-truncation at age 78, and OLS regression estimates for log income. The background characteristics are at the level of parish of birth. All groups divided at the median. Standard errors clustered at the parish of birth level (414 parishes).
P-values in parentheses
*** p<0.01, ** p<0.05, * p<0.1

Table 7 – Robustness analyses for mortality and income in ages 78–95, cohorts 1890–1917 Sweden

	Mortality risk	**Log income**	**Mortality risk**	**Log income**
	A – Correcting for survival bias using Heckman two-stage procedure		*D – Adding parish-specific controls for age structure*	
post X health district	0.940***	0.0200**	0.943***	0.0208**
	(0.006)	(0.044)	(0.009)	(0.024)
Cohort FE	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes
Individuals	39,604	38,618	39,604	38,618
Deaths	36,429		36,429	
Parishes of birth	414	414	414	414
	B – Excluding counties of birth affected by emigration		*E – Adding parish-specific controls for local labour market and migrant structure*	
post X health district	0.938***	0.0186*	0.930***	0.0251**
	(0.006)	(0.081)	(0.004)	(0.016)
Cohort FE	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes
Individuals	36,023	35,134	39,604	38,618
Deaths	33,127		36,429	
Parishes of birth	385	385	414	414
	C - Excluding counties of birth affected by WWI		*F – Excluding counties of residence affected by Spanish flu*	
post X health district	0.933***	0.0248**	0.934***	0.0225**
	(0.004)	(0.018)	(0.005)	(0.043)
Cohort FE	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes
Individuals	35,320	34,453	33,437	31,433
Deaths	32,391		30,609	
Parishes of birth	409	409	411	411

Note: exponentiated coefficients from Cox proportional hazards models for mortality, adjusted for left-truncation at age 78, and OLS regression estimates for log income. Standard errors clustered at the parish of birth level.
P-values in parentheses. *** p<0.01, ** p<0.05, * p<0.1

APPENDICES

Appendix A - Description of the data sources

We gathered data on the division of the parishes into health districts and its changes from several sources. Primarily, governmental reports on provincial doctor districts contain detailed data on the allocation of the parishes as well as the creation dates and funding of the new districts collected from the health board acts (Medicinalstyrelsen 1907, 1939). We additionally verify these divisions with several sources, such as the provincial doctor reports attained from the National Archive in Sweden (Årberättelse 1893–1936), statistical yearbooks on health care (Statistiska Centralbyrån BISOS K, SOS 1880–1917, Statistiska Centralbyrån 1880–1917a) and on public health investment (Statistiska Centralbyrån BISOS U 1874–1917). These sources provided information on the number of the medical personnel employed, such as doctors and midwives, and public spending, both in health care and in education, infrastructure and welfare. In the latter case, we obtained the investment series for each parish before and after the establishment year, and aggregated them to a health district level, which allowed us to carefully determine the intervention dates. All urbanized (*stad*) and semi-urbanized (*köping*) parishes are excluded and therefore the sample comprises only rural parishes (*land*). We also exclude from the analysis the rural parishes that were developed throughout the period into small towns (*köping*), parishes that experienced several health district re-allocations or those where the adoption dates were uncertain (220 out of 2353 parishes). Although the creation of the medical districts continued from 1890 up until 1940s, we stop to follow the establishment of medical districts in 1917.

Data for health districts are gathered from:

1. Statistiska Centralbyrån, (1880–1917). BISOS U: Bidrag till Sveriges officiella statistik U. Kommunernas fattigvård och finanser, Statistiska centralbyrån, P.A. Norstedt & Söner, Stockholm (annual volumes).
2. ———, (1880–1910). BISOS K: Bidrag till Sveriges officiella statistik K. Hälso- och sjukvården. Statistiska centralbyrån, P.A. Norstedt & Söner, Stockholm (annual volumes).
3. ———, (1911–1917). SOS: Sveriges officiella statistik. Allmän om Hälso- och sjukvård. Statistiska centralbyrån, P.A. Norstedt&Söner, Stockholm (annual volumes).
4. Archival sources from the National Archive of Sweden, Medicinalstyrelsen ’Årberättelse från Förste provinsialläkare’ i Malmohus län och Kristianstads län 1881-1946.

Administrative divisions of health districts into parishes are gathered from:

1. Betänkande angående rikets indelning in läkardistrikt samt tjänsteläkarens anställning och åligganden afgigtet af den af Kungl. Maj:t för sådant ändamål tillsatta kommitté, Stockholm, Kungl. Boktryckeriet, P.A.Norstedt & Söner, 1907.
2. Rikets indelning i provinsialläkardistrikt före 1/7 1939 och Medicinalstyrelsens yttrande och förslag till stadsläkarsakuniga den maj 1932, National Arkiv, Sverige, 1939.

Appendix B

Table – Characteristics of the old and newly established provincial health districts

Characteristic	Existed prior to 1880	Established 1881-1917
Number of health districts	163	124
Number of parishes per health district	14,1	6,9
Total population per health district	22,617	11,779
Number of midwives employed	-	4,4
Midwives employed per 10,000 population	-	5,6
Number of (extra or provincial) doctors employed per 10,000 population	-	1,9
Real investments into health care prior, 1900SEK	18,266	16,148
Real investments into health care prior per 1000 population, 1900SEK	715	1,611
Real investments into health care after, 1900SEK	-	19,267
Δ in real investments into health care, 1900SEK	-	4,928
Δ in real investments into health care per 1,000 population, 1900SEK	-	684
Real total public spending prior, 1900SEK	196,182	142,668
Real total public spending prior per 1,000 population, 1900SEK	6,792	11,638
Real total public spending after, 1900SEK	-	130,857
Δ in total public spending, 1900SEK	-	8,317
Δ in total public spending per 1,000 population, 1900SEK		1,272

Note: Parish- and health-district indicators gathered from Statistiska Centralbyrån, BISOS U and K 1880–1917.

Appendix C

Table – Factors of staggered implementation of the reform, 1890–1917

	after1907=1	
	levels 1880	differences 1890 to 1880
log real investment into health care per parish	0.198***	-
	(0.000)	
log real education, infrastructure and welfare spending per parish	0.136***	-
	(0.000)	
log population	-0.0688**	-0.904***
	(0.013)	(0.000)
share of elite and industrial workers in male population 15 – 55 ages	0.298	-0.346
	(0.123)	(0.197)
share of agricultural workers in male population 15 – 55 ages	0.0936	0.558**
	(0.647)	(0.019)
mean age of female	0.0118	0.0216
	(0.291)	(0.146)
share females in total	1.755	2.121
	(0.147)	(0.134)
share in labour force in total 15 – 55 ages	0.567**	0.0105
	(0.010)	(0.970)
share married in total 15 – 55 ages	-0.169	0.0840
	(0.657)	(0.884)
mean family size	-0.212***	-0.0344
	(0.000)	(0.618)
share under age 1 in total	-4.232	-3.656
	(0.291)	(0.269)
share above age 55 in total	0.882	1.939*
	(0.306)	(0.0549)
mortality rate under age 15	0.00239	0.000332
	(0.765)	(0.775)
share (non)disabled	0.0720	3.890
	(0.985)	(0.297)
Railway	0.0533	0.00900
	(0.284)	(0.849)
water supplies improvements	0.216*	0.112
	(0.053)	(0.372)
Observations	492	492

Note: OLS regression estimates. All characteristics are parish-level. The mean implementation year is 1907. Each coefficient is estimated separately. See Appendix for data sources and description. Parish-level indicators gathered from Censuses 1880, 1890, 1900 and 1910, and from Statistiska Centralbyrån, BISOS U and K 1880–1917.
P-values in parentheses.
*** p<0.01, ** p<0.05, * p<0.1

Appendix D

The cohorts born between 1890 and 1917 appear in the SIP dataset from 1968, consistently between the ages 78 and 95. We therefore do not observe individuals that died or migrated from Sweden prior to these ages. In SIP, the data on parish of birth (together with county of birth) are given in text format, and we used an automatic procedure to match these names to parish names in our treatment health district dataset that we further check manually. In the estimation sample, out of 492 parishes treated by the reform, we observe representatives of the 414 parishes.

We gathered information on one-year survivors born in rural areas (live births minus infant deaths) of the cohorts born 1890-1917 from Statistiska Centralbyrån, BISOS A. In Figure we plot them against counts of individuals with rural places of birth available in SIP by cohort and those which have valid information on the parish of birth. Increasing fraction of individuals observed in the SIP dataset compared to a rather stable fraction of one-year survivors from rural places indicate that the individuals born 1890 – 1917 were dying at an increasing rate between the years 1 and 77. As the proportion of the individuals treated by the reform is increasing over time, the effects of the reform can be thus underestimated.

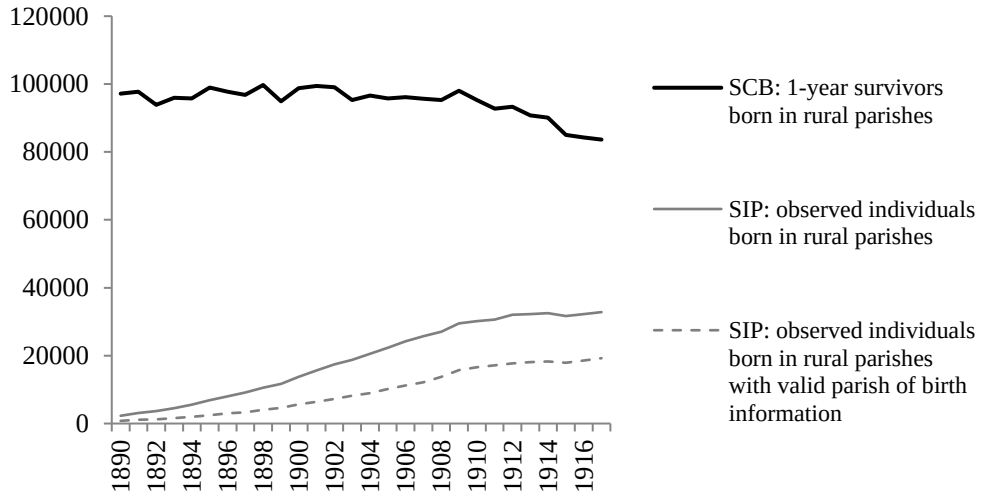


Figure – One-year survivors and estimation sample for the cohorts 1890–1917
Source: SIP and SCB.

Appendix E

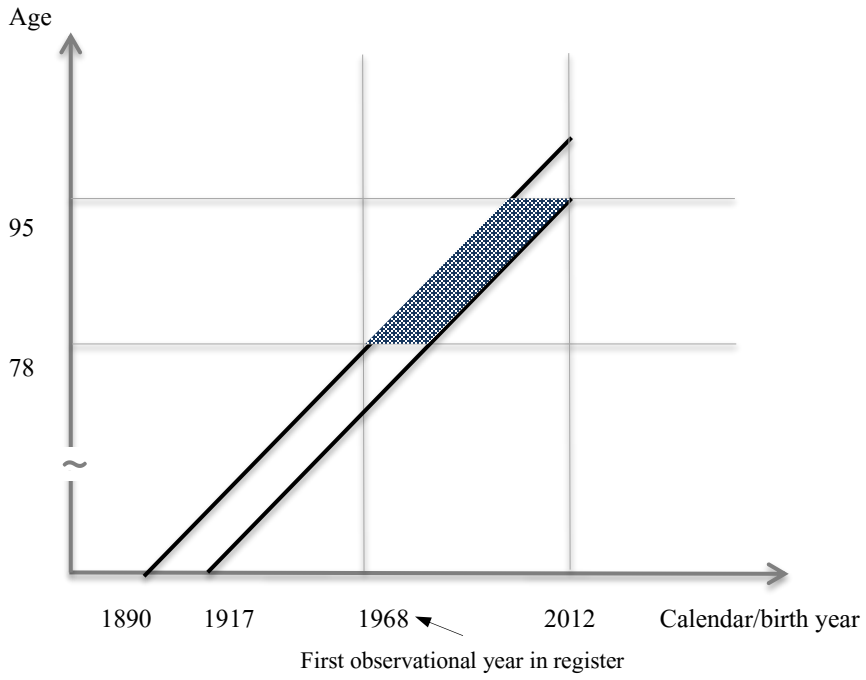


Figure – Lexis chart illustrating the estimation sample

Appendix F – The cause-of-death groups

The death and cause-of-death data is obtained from the Swedish death register and the Swedish cause-of-death register. These registers adopted the different revisions of the international classifications of the causes of death throughout 1968–2012, such as the revision 8 for 1968–1986, the revision 9 for 1987–1996, and the revision 10 for 1997–2012. We classify all causes of death into five groups, such as infectious diseases, cardiovascular diseases, diabetes, cancer, degenerative diseases of tissues and organs, and other causes including violent and ill-defined causes. The exact codes used for these groupings are provided in the following table:

Table – Diagnoses groups across different revisions of the ICD, 1968–2012

	ICD-8	ICD-9	ICD-10
Infectious diseases	000-136; 320-324; 460-519	001-139; 320-324; 460-519	A00-B99; G00-G09; J00-J99
Cardiovascular diseases	390-458	390-459	I00-I99
Diabetes	250	250	E10-E14
Cancer	140-239	140-239	C00-D48
Degenerative diseases	240-246; 251-315; 325-389; 520-789	240-246; 251-319; 325-389; 520-796	D50-E07; E15-F99; G10-H95; K00-R94
Other causes	790-796; E800-Y87	797-999; E800-V82	R95-Z99

Appendix G – Matching procedure

Figure 1 plots a cumulative number of parishes by their dates of the reform implementation. 492 out of 2133 rural parishes established new health districts. From a sample of parishes untreated by the reform we select one-to-one matches to each treated parish, based on the variety of the pre-treatment parish and health-district characteristics at both levels and trends. The motivation for the determinants of health district implementation is a standard model of public health care utilization that involves socio-economic, infrastructure, health, demographic and health system domains. For this approach, we calculate propensity scores and apply a nearest neighbor matching, in which we allow it to find only one control without replacement and impose a common support restriction, with a caliper 0.10. Narrowing or widening of the caliper gives qualitatively analogous results. After the matching, the control and treated groups exhibit a more similar distribution by propensity scores (see Figure 2). As can be seen in Table, the matching procedure allowed us to arrive at 432 treated and 432 matched parishes and therefore substantially enlarge the estimation sample. After the procedure, there are no significant differences between the treated and control groups of parishes by all parish- and health-district level characteristics.

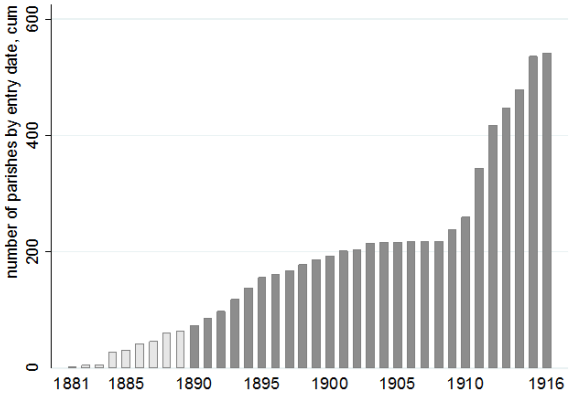


Figure 1 - Number of parishes by their date of entry into treatment, 1881–1917

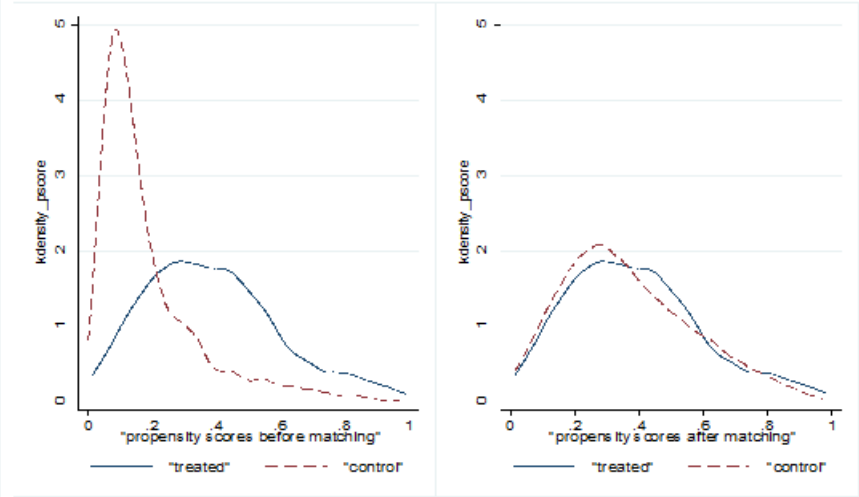


Figure 2 – Density of the parishes of birth over the propensity scores

Table – Parish and health-district pre-treatment characteristics used for the full sample (control and implemented) and matched sample

	Before matching			**After matching**		
Variable	**Control** Mean 1	**Treated** Mean 2	p-value 3	**Control** Mean 4	**Treated** Mean 5	p-value 6
Levels			1 versus 2			5 versus 6
log real health care investment per parish	6,422	7,260	0,000	7,208	7,126	0,153
log real education, infrastructure and welfare spending per parish	8,988	9,402	0,000	9,363	9,330	0,408
log total population 1880	7,130	7,232	0,011	7,277	7,226	0,335
share elite and industrial workers 1880	0,256	0,266	0,045	0,262	0,264	0,731
share agricultural workers 1880	0,421	0,406	0,005	0,410	0,408	0,867
mean age of female 1880	29,295	29,250	0,638	29,241	29,250	0,943
share females 1880	0,510	0,507	0,010	0,507	0,507	0,500
share in labour force 1880	0,674	0,666	0,074	0,666	0,666	0,996
share married 1880	0,480	0,480	0,800	0,483	0,482	0,859
share infants 1880	0,025	0,025	0,706	0,025	0,025	0,573
share older 55 ages 1880	0,150	0,150	0,670	0,150	0,150	0,883
under 15 mortality rate 1880	0,695	0,753	0,617	0,868	0,818	0,788
share (non)disabled 1880	0.992	0.993	0,024	0,992	0,992	0,560
mean family size 1880	4,037	4,015	0,316	4,030	4,025	0,890
railway1880	0,235	0,234	0,968	0,229	0,226	0,935
water supplies 1880	0,045	0,037	0,415	0,035	0,037	0,855
Differences						
diff log total population 1880-1890	-0,027	-0,027	0,792	-0,016	-0,020	0,657
diff share elite and industrial workers 1880-1890	-0,015	-0,009	0,093	-0,004	-0,008	0,408
diff share agricultural workers 1880-1890	0,021	0,013	0,106	0,010	0,014	0,547
diff mean age of female 1880-1890	1,140	1,319	0,017	1,262	1,202	0,561
diff share females 1880-1890	0,001	0,001	0,590	-0,001	0,001	0,312
diff share in labour force 1880-1890	0,023	0,028	0,136	0,028	0,028	0,985
diff share married 1880-1890	0,004	0,002	0,264	0,003	0,003	0,940
diff share infants 1880-1890	-0,001	-0,002	0,223	-0,002	-0,002	0,348
diff share older 55 ages 1880-1890	0,026	0,028	0,070	0,026	0,026	0,994
diff under 15 mortality rate 1880-1890	6,604	6,226	0,629	6,637	6,359	0,811
diff share (non)disabled 1880-1890	-0.001	-0.001	0,369	-0,001	-0,001	0,991
diff mean family size 1880-1890	-0,185	-0,189	0,806	-0,192	-0,183	0,656
diff railway 1880-1890	-0,090	-0,077	0,560	-0,069	-0,085	0,596
diff water supplies 1880-1890	0,010	0,016	0,555	0,019	0,016	0,847
Number of parishes	**1641**	**492**		**432**	**432**	
Number of health districts	**163**	**107**		**131**	**96**	

Note: log real health care investment per parish and log real education, infrastructure and welfare spending per parish are obtained for year 1880 for control parishes and for one year prior to the establishment of a health district for treated parishes.

Appendix H - Additional results for mortality

Table – Robustness analyses. Gompertz proportional hazards model. Effect of the reform on mortality 78–95 ages, cohorts 1890–1917

	I	***I***	***I***	***I***	***M***
post X health district	0.944**	0.945**	0.943*	0.959*	0.957**
p-value	(0.016)	(0.019)	(0.060)	(0.088)	(0.019)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	39,604	39,604	39,604	39,604	69,939
Deaths	36,429	36,429	36,429	36,429	64,451

Note: Models are adjusted for the left-truncation at age 78. *I* denotes a sample of implemented parishes of birth, *M* - sample of implemented and matched. Standard errors clustered at the parish of birth level (414 parishes). For parish-specific trends age interval is 78 – 96. See main text for further description.

Table – Robustness analyses. Effect of the reform on alternative measures of survival 78–95 ages, cohorts 1890–1917

	I	***I***	***I***	***I***	***M***
dead=1, OLS, **78–82 ages**					
post X health district	-0.0187**	-0.0205**	-0.0165	-0.0208**	-0.0117**
p-value	(0.048)	(0.033)	(0.193)	(0.041)	(0.090)
dead=1, OLS, **78–87 ages**					
post X health district	-0.0239**	-0.0230**	-0.0255**	-0.0208*	-0.0201***
p-value	(0.026)	(0.030)	(0.043)	(0.056)	(0.006)
dead=1, OLS, **78–95 ages**					
post X health district	-0.0019	-0.0017	-0.0074	-0.0015	-0.0034
p-value	(0.717)	(0.751)	(0.313)	(0.804)	(0.431)
log time alive, Tobit, **78–95 ages**					
post X health district	0.0769**	0.0761**	0.0572	0.0704***	0.0466**
p-value	(0.014)	(0.017)	(0.128)	(0.000)	(0.037)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	39,604	39,604	39,604	39,604	69,939

Note: *I* denotes a sample of implemented parishes of birth, *M* - sample of implemented and matched. Standard errors clustered at the parish of birth level (414 parishes). See main text for further description. For the tobit models, the observations are right-censored at 2.890 (log of 18 years). Mean in not-treated is 9.4 years.

Appendix I - Additional results for log income

Table – Robustness analyses. OLS models. Effect of the reform on permanent income 78–95 ages, cohorts 1890–1917

	I	***I***	***I***	***I***	***M***
life-time earnings 78–95 ages					
post X health district	0.0199**	0.0217**	0.0196**	0.0238**	0.0215***
p-value	(0.037)	(0.032)	(0.045)	(0.027)	(0.007)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,618	38,618	38,618	38,618	68,224
log income 78–82 ages					
post X health district	0.0222**	0.0246**	0.0261*	0.0236**	0.0193**
p-value	(0.028)	(0.024)	(0.019)	(0.041)	(0.017)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,609	38,609	38,609	38,609	68,208
log income 78–87 ages					
post X health district	0.0208**	0.0214**	0.0227**	0.0245**	0.0178**
p-value	(0.035)	(0.040)	(0.029)	(0.034)	(0.021)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes	yes	yes	
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,615	38,615	38,615	38,615	68,218
log income 78–95 ages, closest value for income instead of the null					
post X health district	0.0156*	0.0161*	0.0218**	0.0210**	0.0150**
p-value	(0.066)	(0.074)	(0.017)	(0.040)	(0.029)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	38,618	38,618	38,619	38,619	68,224
log income 78–95 ages, Excluding years under widowhood					
post X health district	0.0225*	0.0241*	0.0261*	0.0307**	0.0235**
p-value	(0.086)	(0.080)	(0.065)	(0.045)	(0.013)
Cohort FE	yes	yes	yes	yes	yes
Parish of birth FE	yes	yes	yes	yes	yes
County of birth x cohort linear trends		yes			
Parish of birth Xs x cohort FE			yes		
Parish of birth x cohort linear trends				yes	
Individuals	27,824	27,824	27,824	27,824	48,941

Appendix K – Robustness analyses for selective survival

Table – Robustness analyses. Effect of the reform on share of survivors, 1890–1917

	(1) 1890-1917	(2) 1890-1906	(3) 1907-1917
post X health district	0.0268**	0.0462***	0.0179
	(0.032)	(0.001)	(0.288)
mean of the outcome	0.388	0.239	0.484
Cohort FE	yes	yes	yes
Parish of birth FE	yes	yes	yes
Observations	3,278	1,170	2,108
R-sq	0.063	0.174	0.016
Number of parishes	414	250	281

Note: The analyses restricted to parish-cohorts used in the sample. Data on number of infants (those below age 2 to avoid differences in the timing in enumeration across parishes) is obtained from censuses 1880–1910 at a parish-of-birthXcohort level, and data on the number of individuals survived to the age 78 is from SIP.

Appendix L - Additional robustness analyses for selective migration and fertility

Table – Robustness analyses. Effect of the reform on migrant structure and household size of the parishes of birth, 1890–1917

	(1) Share of migrants	(2) Household size
post X health district	0.00100	0.00141
	(0.372)	(0.851)
Cohort FE	yes	yes
Parish of birth FE	yes	yes
Observations	3,278	3,278
R-squared	0.082	0.006
Number of parishes	414	414

Note: The analyses restricted to parish-cohorts used in the sample. Data is obtained from censuses 1880–1910 and recalculated to parish-of-birthXcohort level.